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Short Communication

Hybrid is not a dirty word: Commentary on Wade and Kazeck (2017)

Peter H. Wilson [a,b,*], Karen Caeyenberghs [a,b], Deborah Dewey [c], Bouwien Smits-Engelsman [d], Bert Steenbergen [a,b,e]

[a] School of Psychology | Faculty of Health Sciences, Australian Catholic University, Melbourne, Australia
[b] Centre for Disability and Development Research (CeDDR), Australian Catholic University, Australia
[c] Departments of Pediatrics and Community Health Sciences, and Owerko Centre, Alberta Children's Hospital Research Institute (ACHRI), University of Calgary, Canada
[d] Department of Health and Rehabilitation Sciences, Faculty of Health Sciences, University of Cape Town, South Africa
[e] Behavioural Science Institute, Radboud University Nijmegen, The Netherlands

The review by Wade presents some provocative arguments on the state of research and hypotheses on the possible cause (s) of developmental coordination disorder (DCD). He contends that degrading of perception-action coupling (i.e., dynamical systems approach) better explains the motor deficits seen in children with DCD than problems in internal modelling (i.e., information processing approach). However, Wade's assessment lacks an appreciation of how experimental work on DCD has progressed over the past 10–15 years, moving beyond the narrow box-and-arrow models of traditional information processing (IP) theory and into the realm of cognitive neuroscience. Indeed, Wade's critique overlooks a large body of recent work that would help dispel many of the misgivings that he presents in his paper. The following commentary will highlight what we consider are the main points in defence of the cognitive neuroscientific investigation of DCD. We will also discuss areas of weakness in present theoretical models and how ecological principles gleaned from a dynamical systems approach could inform and enrich our understanding of the underlying cause(s) of DCD going forward.

1. A mid-century straw man (or Mr. Schema does not live here anymore)

The recent body of experimental work on DCD has largely moved beyond the narrow confines of traditional IP theory. Wade presents, however, a straw man stuffed with concepts that few today would consider valid representations of the motor system: *motor schema*, *motor program*, "sense-think-act cycle", etc. are presented as being synonymous with current thinking. In actual fact, constructs like *motor program* for example have not been a focus of DCD research for 20 years or more. Such constructs carry with them a lot of conceptual baggage, including the idea that processing occurs in a linear fashion from input to output. Few present-day researchers would subscribe to this view, and yet Wade argues that thinking in the DCD field is still constrained by it. This position is supported by a selective and limited critique of the actual work that has been conducted on DCD, over the last decade. Since our 2012 *meta*-analysis (Wilson, Ruddock, Smits-Engelsman, Polatajko, & Blank, 2013) there have been well over 100 experimental studies on DCD, and nearly 20 neuroimaging studies, alone. Wade's review cites 5 individual studies conducted after 2009 (two his own), and only one study published after 2012 (an excellent paper by Wilmut, Du, and Barnett (2015)). This is not an informed platform on which to base this stinging critique.

Without doubt, the field of cognitive neuroscience (and its various offshoots) presents a stark contrast with traditional IP theory. Not only has the discourse moved to understanding brain-behaviour relations but also to how the brain is modified in a reciprocal manner by the environment and experience, whether over short timescales or longer (e.g., Koziol & et al., 2014),

* Corresponding author at: School of Psychology, and Centre for Disability and Development Research (CeDDR), Australian Catholic University, 115 Victoria Parade, Melbourne, Victoria 3065, Australia.
E-mail address: peterh.wilson@acu.edu.au (P.H. Wilson).

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and how an individual's genetic makeup can influence brains and behaviour (Mosca & et al., 2016). This more refined version of cognitive neuroscience also implies a synergistic relationship between the individual performer and the environment, even if the methods are generally focused on a tightly controlled set of parameters (viz task constraints). Moreover, notions of modularity in the structure of the brain have given way to interactive network models (e.g., Sripada, Kessler, & Angstadt, 2016) and developmental theories like *interactive specialisation* (Johnson, 2011). These theoretical advances have in fact broadened the view of cognition and action as an embodied phenomenon. On the importance of this development in knowledge and understanding, we agree with Wade. Unlike Wade, we do not draw a distinction between notions of embodied cognition and general trends in the experimental investigation of DCD over recent years.

The notion of *embodied cognition* (EC) is not specific to traditional ecological theory (despite what is implied by Wade), but rather is an important notion in modern conceptions of thought and action from a broad cognitive science perspective. The very notion of EC acknowledges the centrality of the physical system in development and learning, even with respect to how higher-order knowledge constructs are developed through experience (Gallese & Lakoff, 2005). In the case of motor behaviour, the quest to model these basic biological systems in action is not an act of surrender to reductionist logic. We would agree that by the deft hand of evolution, action systems are exquisitely attuned to information from the ambient environment (and not "meaningless inputs" to quote Wade); these basic systems bias processing in ways that serve action goals and survival. We are intimately intrigued by this notion, and seek to identify and understand the structure (and function) of these basic neuromotor systems, how they change with maturation, and at the same time are shaped by the environment. Whether called mechanistic or not, this quest is important to fully understand typical motor development, developmental delays, and significant departures from typical growth patterns (Ruddock et al., 2016).

We concede that the part of the equation that has remained elusive in studies of DCD concerns the dynamic interplay between movement, experience and developing brain systems. We are starting to see some inroads into this dynamic in other fields that study structural neuroplasticity in response to training. These longitudinal magnetic resonance imaging (MRI) training studies examine learning-induced alterations in functionally relevant brain areas and interconnected structural networks in relationship with improvements in motor performance. In healthy adults, experience-dependent changes following motor skill training have been found in microstructure and macrostructure when triggered with only relatively short periods (in terms of weeks) of motor practice (Taubert & et al., 2010). Moreover, there is increasing evidence of plastic changes in brain structure in children as well. For example, Drijkoningen et al. (2015) has applied advanced diffusion MRI technology to sensitively track neuroplastic alterations in the cerebellar peduncles using PC-based portable balance control training programs (for two months) in young patients (aged 8 to 20 years) with traumatic brain injury and an age-matched healthy control group (Drijkoningen et al., 2015). Results revealed cerebellar white matter changes and improvements in dynamic aspects of postural control in both groups. A similar approach for enhancing structural neuroplasticity and motor control could also be implemented in children with DCD. One approach is to examine learning and development parametrically by looking at the dynamics of the interaction between task, context and the neuromotor system – a "hybrid approach"!

2. Perceptual-motor coupling and coordination

Wade frequently defers to the basic hypothesis that DCD is explained by a deficit in "perceptual-motor coupling" or "in the perception-action relationship". This view, in fact, is more a global description of the kinematics of the condition than a causal explanation. It remains unclear exactly what this "hypothesis" means in terms of causality, or indeed, possible mechanisms even at the level of person-environment interactions. It remains the case that the arguments presented by Wade in this review do not extend much beyond a descriptive level of analysis. For example, possible mechanisms of perceptual-motor coupling are not entertained. Maturation and development of the neuromotor system itself must be a part of any such explanation, conceived of as an Individual level constraint on motor behaviour. Indeed, this may be a point of possible integration between explanatory frameworks. It is perhaps ironic that one of most important components of the individual system – the brain – rarely features in any ecological account of DCD. Rather, traditional ecological/dynamical accounts are largely concerned with the changing dynamics (both kinematic and kinetic) of the effector systems as a function of variations in task-environmental constraints; e.g., changes in rhythmic auditory-motor coupling as a function of the stimulus beat. On occasion, dynamical processes of this type are discussed in relation to neurocognitive mechanisms (Whitall et al., 2008).

The notions of *self-organisation* and *direct perception* are important and powerful postulates of the ecological approach but should not be immune to questions about mechanisms. If (causal) ecological accounts assert that DCD reflects a reduced ability to judge potential actions in relation to affordances or perhaps reduced coupling, the trick is to then work out why and how. For many children with DCD, development of efficient motor coordination and adequate levels of skill are elusive, despite ample experience or practice. Moreover, even basic processes of motor learning that are largely implicit have been shown to be reduced in DCD (Steenbergen, van der Kamp, Verneau, Jongbloed-Pereboom, & Masters, 2010). In our 2012 *meta*-analysis (Wilson et al., 2013) and an updated systematic review of work conducted between mid-2011 and September 2016, effect sizes for group (DCD vs. non-DCD) were large across studies, and across different paradigms. These effects cannot be ignored. Mapping possible underlying mechanism within a developmental framework is especially complex as the organism is constantly maturing and changing with experience—this is not reducible to box-and-arrow logic. The changing

interaction of individual (cognitive-motor) and environmental constraints as a function of genes, brains, age and experience will need to be examined in order to identify the underlying causes of DCD (Dewey and Bernier, 2016; Ruddock et al., 2016).

3. Converging lines of evidence

Converging evidence (or *consilience*) provides a strong basis upon which to draw conclusions (Wilson & McKenzie, 1998). Recent reviews highlight the substantial growth in experimental research on DCD, now at a rate of around two new published studies every month (Wilson et al., under revision). The lines of convergence in this research in relation to internal modelling (Wade's whipping boy), the mirror neuron system (MNS) (Reynolds et al., 2015) and neural network analyses (Caeyenberghs et al., 2016) cannot be ignored. In the particular case of the Internal Modelling Deficit (IMD) hypothesis, evidence is drawn from target-directed reaching, postural control, motor imagery, oculomotor control, and attentional control, suggesting that the scope of this hypothesis holds up well across effector systems and task complexity (Adams, Lust, Wilson, & Steenbergen, 2016): children with DCD have difficulty using forward estimates of limb/eye position as a control parameter when performing a skill or even maintaining a stable body position. In our 2012 review, well over 200 effect sizes were presented on IMD-related functions across more than 30 studies, with large group effects for most categories (Cohen's $d_w > 1.0$), and several major categories >1.5. Proponents of the IMD hypothesis have never asserted that it is "the sole valid theoretical position". It continues, however, to spawn a range of new and innovative studies from a cognitive neuroscience perspective. Wade's highly selective review fails to capture this fact.

4. Does DCD have a biological basis? Neural substrate and genetics

Wade's claim that DCD has no brain-based/biological origins is at odds with accumulating evidence using a wide array of neuroimaging techniques including magnetic resonance imaging (MRI), electroencephalography (EEG), and transcranial ultrasound (Wilson et al., under revision). Neuroimaging studies reveal that children with DCD show modified brain activation patterns reflecting disruptions of white matter connections. Just because DSM-5 states that DCD occurs in children with no known neurological disorders, does not mean that changes in brain structure and function at the group level do not exist, identified using well-established and state of the art MRI scan sequences (not 'obtrusive technology', as asserted by Wade). Of note, the dichotomization (cerebellum versus basal ganglia group) that Wade mentions is not at all representative of the neuroimaging literature—this sub-division is used rarely in the literature. While we suggest that neuroimaging results be interpreted with caution (e.g. relatively small sample size multiple comparisons problems), these studies show a strong association between motor deficits and altered structural and functional properties of the brain of children with DCD.

Recently, the MRI literature has moved away from a localizationist view of the brain ("focusing on an array of functions in the brain") and begun to examine the developing brain as a network structure (or 'connectome') using a graph theoretical approach. For example, Debrabant et al. (2015) revealed a weaker segregation and integration of the structural network in children with DCD, and a significant relationship between these abnormalities of the connectome and visuomotor deficits. We acknowledge that the underlying biological mechanisms of the neural changes are still unknown and further work is needed before neuroimaging metrics may be implemented as clinically relevant biomarkers that could guide rehabilitation efforts in DCD. However, the domain of DCD has a body of brain-based evidence that can no longer be ignored.

Besides alterations in brain structure and function, DCD also appears to have a genetic component. Initial studies showed that DCD is highly heritable with estimates approaching 70% (Lichtenstein, Carlström, Råstam, Gillberg, & Anckarsäter, 2010; Martin, Piek, & Hay 2006), suggesting that DCD is in part caused by genetic or genomic variation. A more recent study that investigated copy number variation (CNV) – structural variation that is the result of deletions or duplications in the genome – revealed an increased rate of rare CNVs and rare, genic CNVs in children with DCD (Mosca et al., 2016). In the genic CNVs, there was enrichment for brain-expressed genes and genes previously implicated in other neurodevelopmental disorders such as attention-deficit/hyperactivity disorder, autism spectrum disorder and Tourette syndrome. Genes and loci of particular interest included GAP43, RBFOX1, PTPRN2, SHANK3, 16p11.2 and distal 22q11.2, all of which have been linked with disruptions in brain function and neurobehaviour. These findings provide strong support that specific genes and/or gene networks are implicated in the development and function of neural circuits associated with DCD, and provide compelling evidence supporting a genetic basis for DCD. In sum, brain alterations and genes are likely to be causes of DCD that result in executive function (EF) and IP deficits, as discussed in the next sections.

5. Executive function (EF)

Deficits in EF are not trivial to our understanding of DCD! Difficulties are evident across all major domains: working memory, both visuospatial and verbal ($d = 1.07$), inhibitory control (1.03), and executive attention (1.46)—see Wilson et al. (2013). Our recent review included 16 additional studies that showed strong effects sizes on measures of hot EF[1] and everyday EF which support self-regulation and organisation. Moreover, persistent difficulties in EF have been reported

[1] Hot EF refers to those cognitive control functions that are enlisted when the performer plans actions in accordance with potential motivational/emotional rewards and costs.

in individuals with DCD from childhood into early adulthood. These difficulties have implications for participation in different life contexts, e.g. managing life transitions into higher education and occupational performance (Kirby, Edwards, & Sugden, 2010). Wade fails to represent this work in his review.

So prominent are EF deficits in DCD that some have suggested that the presence of DCD and executive dysfunction is more the rule than the exception (Martin et al., 2006). However, longitudinal work is needed to unpack the co-relationship between motor control and cognition in DCD. On recent example is a study by Ruddock et al. (2016) showing poor integration between inhibitory control and the online control of movement.

6. Hybrid is not a dirty word

Perhaps one of the longest running debates in philosophy concerns that between nativism and empiricism. But more recent are calls for an approach to understanding development that embraces both "innate predispositions" and constructivism (Karmiloff-Smith, 2009)—call it a hybrid perspective. At no time in the history of work on DCD is this more relevant.

One of the most important principles of motor behaviour is the notion that behaviour is the product of an interaction between the individual agent, the task, and the environment. Again, we share this notion and advocate for a hybrid position, one where a variety of methods (including genetics, neuroimaging, kinematic/kinetic analyses, and behavioural experiments) can be enlisted to help identify key constraints at the individual level (some shared across children and others more idiosyncractic). The integration of ecological principles shows us the importance of identifying how individual-level constraints operate as a function of task and environmental context. This is shown by recent work (that can be described loosely as *constraints testing*) where children with DCD display compensatory strategies that may be a response to poorly developed motor control systems at the individual level (like anticipatory control). For example, the work of Wilmut et al. (2015) provides an excellent model for understanding how navigation through apertures can be understood as a process of selecting an adaptive movement strategy that takes into account the level of functional skill available and the need to avoid collision. The approach does not suggest that the navigation path is specified directly to all, but that for those adults with DCD there was some recognition about the limits of the control, which necessitated a greater safety margin when approaching an aperture. Similar compensatory adjustments have been seen in catching and reach-to-grasp actions where safety margins for grip aperture are larger and occur earlier in flight in DCD. These data are interpreted in terms of computational motor control, but would benefit from a systematic investigation of interactive constraints over the course of child development.

7. Constraint, condition or task parameter: Are we talking about similar things?

Dynamicists frequently talk about the impact of varying *constraints* on perormance; e.g., the effect of the length of a stick on manual wielding (Wade, Tsai, Stoffrege, Chang, & Chen, 2016). Use of the term, *constraint*, is no longer the reserve of dynamical systems theory (even though it may have originated from within that framework). Many of the experimental (task) conditions that are enlisted to dissect and understand the performance patterns of children with DCD are not far removed from the constraints testing that we see in ecological research. Perhaps what differs most is the interpretation. What is a variation in task condition for one researcher might be a variation in task constraints for another, and sometimes the two terms are used interchangeably. Wade suggests, "children diagnosed with DCD may be exhibiting a deficit associated with a constraint in fully detecting relevant perceptual information extant in the environment". The critical thing is whether such manipulations help us better understand the phenomenon of interest—poorly developed movement skill in children.

So what have we learned about the impact of task constraints in recent research? A recently submitted systematic review has shown a number of important themes in this work that relate directly to this question (Wilson et al., under revision): the differential effect of task complexity in terms of spatial, temporal, biomechanical and other demands; the different effect of task constraints on explicit and implicit learning; the ability of children with DCD to compensate for their motor control difficulties, adopting different movement patterns to achieve a task goal. In short, only a very systematic investigation of constraints – whether from a dynamical or cognitive neuroscience perspective – will advance our theorising in DCD.

8. Unpacking variability

We would accept that variability is not well explained in current accounts of DCD, whether used in reference to performance per se or to the parameters of motor control and learning. Generally speaking, variability is construed in negative terms, taken to mean "less consistent" or error ridden. Aspects of so called "good variability" are not well explicated, as Wade highlights. Random (or stochastic) variability is important to ensure that the performer maintains flexibility in the achievement of movement goals despite variations in the ambient environment, especially those that cannot be predicted (Priplata et al., 2005).

This underscores one of the more pertinent criticisms of many kinematic analyses of movement in DCD that appear in the literature—the imprecise explanation of motor variability. Over repeated trials or movement sequences, kinematic parameters are often described as showing more variability, both within-limb and between-limb. These forms of variability are seen as "bad" and confirm that children with DCD find it more difficult to control the multiple degrees of freedom inherent to the

action over repeated trials/cycles. Beyond this, however, explanations of the mechanisms underlying the source of variability are often bereft of detail.

9. Is "noise a useful metaphor to describe causal processes?

In a similar vein, explanations about the role of noise in motor control and learning need further development. Smits-Engelsman and Wilson (2013) discussed the notion of noise in their 2013 review of DCD. Noise was largely conceived of as signal error at various levels of the perceptual-motor system. A limitation was that their conceptualisation did not consider stochastic variability, as Wade points out.

Patterns of neural recruitment with motor learning provide some insight into how "noise" might be better conceived, at least at the brain level. In the case of typical skill learning, we see a gradual refinement in neural recruitment and kinematic efficiency over learning trials. The firing pattern, for example, tends to become more streamlined (or less diffuse). And for tasks with added cognitive demands, the coupling between planning and execution centres shows more streamlined activation (shown by less diffuse activation patterns), less conscious effort, and associated improvements in functional performance. One might be tempted to say that these efficiencies in neural activation and biomechanics over learning trials are the counterpart of "noise". In DCD (or atypical motor development and learning), we hypothesise that these neural couplings are less well developed, but we simply do not have the data to make firm conclusions about these changes with learning. Studies of different sources of variability (i.e., anticipatory and feedback related signals, brain and muscle activations) and the frequency of these signals are needed—see Speedtsberg et al. (2017) for a recent example of work on DCD where variability in postural sway is decomposed into random and error correction components. However, there is a precedent that supports this hypothesis in the literature on disconnection syndromes where noisy (or reduced) functional and structural connectivity is construed as a basis for aberrant behaviour or function.

10. The truth is out there, . . .somewhere in the middle

In conclusion, the critical review of Wade reinforces a sobering reality: the tension between ecological and cognitive neuroscience approaches is not yet reconciled in the study of DCD. Indeed, defining these positions as a dichotomy may not serve us well. Notwithstanding the fact that Wade's take on the experimental literature is selective, it does serve to highlight areas where current (cognitive neuroscience) approaches do fall short (e.g., the resolving variability and the concept of system noise). Somewhere in the middle there is fertile ground for researchers where person-environment interactions can be unravelled using new tools. Mixed methods and fine-grained longitudinal designs (using microgenetic methods for example), are areas well worth investing in by the next generation of researchers as they build a more comprehensive approach to DCD research. Some of the next wave of research can be seen in the constraints testing work of Wilmut et al. (2015), and advances in neuroimaging using connectomics. Whether we call this next wave a hybrid approach or something else does not really matter.

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